

Silicosis and Asbestosis

BY VARIOUS AUTHORS

EDITED BY

A. J. LANZA, M.D.

ASSISTANT MEDICAL DIRECTOR, METROPOLITAN LIFE INSURANCE COMPANY;
CHAIRMAN, INDUSTRIAL HYGIENE COMMITTEE OF THE NEW YORK
TUBERCULOSIS AND HEALTH ASSOCIATION

OXFORD UNIVERSITY PRESS

LONDON NEW YORK TORONTO

Copyright, 1938, by Oxford University Press, New York, Inc.

Printed in the United States of America

PREFACE

THE industrial age has complicated the scheme of life in many ways, some of them directly affecting the public health ; among these are the diseases due to the inhalation of certain kinds of atmospheric dusts arising directly from industrial operations. Ill effects due to dust contamination of the atmosphere have been recognized from earliest times.

This book has for its purpose the presentation of the medical and public health aspects of silicosis and asbestosis, two definitely industrial dust diseases. Silicosis looms larger in the picture than asbestosis because the former is much more widely spread, affects a much greater number of the industrial population, has been recognized and studied longer and, consequently, more is known about it.

The form of the generic term pneumoconiosis, rather than pneumonoconiosis or pneumonokoniosis, has been adhered to throughout the book. The International Labour Office and both British and American editors have adopted it as preferable.

The editor wishes to express special thanks to Miss C. M. Bresnan for reading proof and for making the index and to acknowledge with gratitude the help received from many sources and from many individuals. To them and particularly those working in the field of industrial hygiene, this book is dedicated.

A. J. LANZA, M.D.

February 24, 1938

CONTRIBUTORS

A. J. LANZA, M.D.

Assistant Medical Director, Metropolitan Life Insurance Company ; Chairman, Industrial Hygiene Committee of the New York Tuberculosis and Health Association.

Formerly in Charge of the Office of Industrial Hygiene, United States Public Health Service ; Chief Surgeon of the United States Bureau of Mines ; Adviser on Industrial Hygiene to Commonwealth Government of Australia ; Special Staff Member of the International Health Board of the Rockefeller Foundation ; Medical Consultant of General Motors Corporation.

R. R. SAYERS, A.M.,M.D.

Senior Surgeon, United States Public Health Service ; Chief, Division of Industrial Hygiene, National Institute of Health. Formerly Chief Surgeon, United States Bureau of Mines.

EUGENE P. PENDERGRASS, M.D.

Professor of Radiology, Medical School, University of Pennsylvania ; Professor of Radiology, Graduate School of Medicine, University of Pennsylvania ; Associate Director, Department of Radiology, Hospital of the University of Pennsylvania.

S. ROODHOUSE GLOYNE, M.D.,D.P.H.

Pathologist, Pathological Laboratories and Research Institute, The London Chest Hospital.

LEROY U. GARDNER, M.D.

Director, Saranac Laboratory for the Study of Tuberculosis.

E. L. MIDDLETON, M.D.,D.P.H.

H. M. Medical Inspector of Factories, London, England.

INTRODUCTION

PATHOLOGICAL conditions of the lungs, due to the inhalation of excessive quantities of dust in certain occupations, have been known to exist for many centuries. It is also true that the frequency of infection, particularly pulmonary tuberculosis, as a complication has been realized for a long time. Pneumoconiosis is a comprehensive term, designating the effects upon the lungs of the inhalation of excessive quantities of dust, manifested by structural changes in the lung tissue and entirely distinct from the action of poisonous dust, such as lead or mercury, in which case the lungs act merely as the point of entrance into the body without definite local influence. At first it was associated chiefly with mining but with the advancement of the industrial age, dusty industries multiplied.

It is customary to speak of the dust inhalation effect upon the lung tissue by the name of the chief constituent of the dust breathed, as silicosis when silicon dioxide (SiO_2) is the principal ingredient and asbestosis when asbestos is the offending agent. Silica dust, when the particles are sufficiently small, has the peculiar property of inducing a fibrosis of the lung tissue. This effect is characteristic, as is the resulting roentgenologic appearance. In asbestosis, the pathological process is quite different as is the roentgenologic appearance. These matters are thoroughly dealt with in the text. There is still, however, much to be learned with respect to both conditions. We know the results of the inhalation of these dusts but we are not sure how they are produced.

There are two ways in which the pulmonary infections, tuberculosis and pneumonia, may affect or complicate pneumoconiosis. First, there may be infection before the exposure

to dust. It is an undecided question whether such infection can so change the pulmonary structure that the later dust effect is more pronounced than it would otherwise be. There seems to be reason to believe that such an enhancement is to be expected, especially if the previous infection were tuberculosis, but this must be left to the histopathologist for final proof. Secondly, do such infections upon an already dusted lung become more severe or aggravate the dust effect or otherwise change its character? There is even more evidence that any of these effects may result but here again the histopathologist must be called upon to tell us the exact truth. The first intimation that occurred to us that the character of a silicotic lesion could be so changed was when we were searching for an explanation as to why certain individuals with silicosis showed massive consolidations of fibrosis in the lungs and others in the same occupation and apparently exposed to the same amount of dust of the same variety showed a more evenly diffused nodular fibrotic process. It was a great satisfaction to hear a well known pathologist say that a comparatively recent infection like lobar pneumonia might be the answer but that at present there was no definite proof of such theory being correct.

It was gratifying to learn that most of us were thinking along similar lines. That most cases of silicosis die of an intercurrent infection, especially tuberculosis and possibly pneumonia, is evidence that silicosis may exert some effect upon the progress of some pulmonary infections. It has not been proven as yet that the same is true of asbestosis.

A comprehensive knowledge of the effects upon the lungs of excessive inhalations of dust is not to be quickly gained. The combined efforts of the histologist, pathologist, clinician, roentgenologist, chemist, physicist and other technological experts are necessary. The legal profession deserves a certain share of commendation for stimulating the study of

occupational dust diseases and has made a thorough knowledge of pneumoconiosis a necessity among many of the medical men. Were it not for the demand for exactness from outside the medical profession, it is difficult to say how soon curiosity or medical research would have achieved our present accuracy of knowledge with respect to the etiology, symptoms, and pathology of pulmonary dust diseases and their modification by certain infections.

A large number of us have, no doubt, taken up the study of pneumoconiosis from the standpoint of interest. Following the experience with silicosis in South Africa, starting several decades ago, text books for the medical student simply mentioned it in a more or less cursory manner as an uncommon condition. We are sure of the necessity of many angles of approach to the subject. The first comprehensive report of silicosis in the United States was made about twenty years ago by one of the authors. It described what was occurring among miners in the south west. Almost simultaneously and without any knowledge of this report, it occurred to a small group of medical men in the east that the best way to study the differences in the appearances of trunk shadows in the roentgenograms of the lungs might be to study the chests of persons engaged in various dusty occupations. The main point learned in this investigation was that organic dusts do not induce lung changes but it further stimulated the study of the effects of inorganic dusts, particularly silica, and later, asbestos. This study has continued up to the present time.

Some of the authors of this book were among the pioneers in silicosis and asbestosis ; they have blazed the trail for future investigations. It can be truthfully stated that those who studied these occupational diseases in North America and the British Empire have always had the good of the affected workers in mind. Their researches have saved life and have made many occupations safer for the worker.

HENRY K. PANCOAST, M.D.

CONTENTS

I. HISTORY OF SILICOSIS AND ASBESTOSIS	3
1. History of Silicosis	3
<i>South Africa</i>	6
<i>Great Britain</i>	8
<i>Germany</i>	12
<i>Australia</i>	14
<i>Italy</i>	16
<i>Canada</i>	17
<i>United States</i>	18
2. History of Asbestosis	22
II. ETIOLOGY, SYMPTOMS, DIAGNOSIS OF SILICOSIS AND ASBESTOSIS	31
1. Etiology, Symptoms, and Diagnosis of Silicosis	31
<i>Silica in Nature</i>	32
<i>Occupational Exposure to Silica</i>	33

<i>Factors Influencing the Action of Silica Dust Particles as the Exciting Cause of Silicosis</i>	36
<i>Occupational History</i>	41
<i>Predisposing Causes</i>	47
<i>Symptoms</i>	47
<i>Subjective Symptoms</i>	48
<i>Objective Symptoms</i>	50
<i>Diagnosis</i>	53
<i>The Relation of Silicosis to Disability</i>	55
<i>Prognosis</i>	57
2. Etiology, Symptoms, and Diagnosis of Asbestosis	57
<i>Symptoms</i>	60
<i>Asbestosis Bodies</i>	60
<i>Diagnosis</i>	61
III. ROENTGEN-RAY DIAGNOSIS	66
The Healthy Chest	67
<i>The Soft Parts</i>	67

CONTENTS	ix
<i>The Bones</i>	68
<i>The Pleura</i>	70
<i>The Mediastinum</i>	73
<i>The Diaphragm</i>	80
<i>The Hilum Shadows</i>	82
<i>The Zones</i>	86
<i>The Lobes</i>	86
<i>The Vascular Markings</i>	87
<i>The Bronchi</i>	91
<i>The Lung Fields</i>	92
<i>Effect of the Phases of Respiration on the Roentgen Appearances in the Chest</i>	97
Roentgen Technic	102
Roentgenological Stages of Progress	105
<i>First Stage</i>	106
<i>Second Stage</i>	107
<i>Third Stage</i>	110
<i>Other Classifications</i>	115
<i>Simple Silicosis</i>	117

<i>Tuberculosis with Silicosis</i>	115
<i>Healthy Lungs and Adnexa</i>	124
<i>Simple Silicosis</i>	125
<i>Silicosis with Infection</i>	128
Roentgenologic Considerations of Silicosis and Silicosis with Infection	132
<i>Silicosis</i>	132
<i>Differential Diagnosis</i>	134
<i>Metastatic Malignant Conditions of the Lungs</i>	134
<i>Tuberculosis</i>	134
<i>Mycotic Infections</i>	135
<i>Miliary Calcifications</i>	136
<i>Baritosis</i>	139
<i>Silicosis with Infection (nodular predominance type)</i>	139
<i>Silicosis with Healed Infection (conglomerate nodular type)</i>	144
<i>Silicosis with Active Infection (conglomerate nodular type)</i>	145

<i>Differential Diagnosis</i>	145
<i>Infiltrating or Permeating</i>	
<i>Malignant Metastases</i>	145
<i>Polycythemia or Erythremia</i>	148
<i>Silicosis with Infection and</i>	
<i>Massive Lesions</i>	149
Further Considerations of	
Tuberculosis and Silicosis	151
Complications in Silicosis	161
<i>Primary Bronchogenic Carcinoma</i>	161
<i>Cardiac Lesions</i>	165
The Erythrocyte Sedimentation	
Reaction in Silicosis	166
Asbestosis	166
<i>The Asbestos Industry</i>	166
<i>Autopsy</i>	168
<i>Symptomatology</i>	170
<i>Roentgenologic Considerations</i>	172
<i>Discussion of the Nature of the</i>	
<i>Roentgenologic Appearances seen</i>	
<i>in Asbestosis</i>	176
<i>Prognosis</i>	185

<i>Predisposition to Tuberculosis</i>	187
<i>Differential Diagnosis</i>	
Passive Congestion of the Lungs as a Result of Cardiac Decompensation	188
<i>Advanced Bilateral Bronchiectasis</i>	190
 IV. PATHOLOGY	 198
Introduction	198
<i>Definitions</i>	198
<i>Nature of the Dust</i>	199
<i>The Portal of Entry</i>	201
<i>The Tissue Reaction</i>	202
Silicosis	205
<i>Macroscopical Appearances</i>	205
Lungs and Pleurae	205
<i>Microscopical Appearances</i>	210
The Silicotic Nodule	210
<i>The Complications and Sequelae of Silicosis</i>	214
<i>Silicosis and Tuberculosis</i>	216

CONTENTS	xiii
Silicosis with Obsolescent Tuberculosis	217
Silicosis with Manifest Tuberculosis	218
Tuberculo-Silicosis	218
<i>Some Unusual Pathological Varieties of Silicosis</i>	221
Silicosis in Coal Miners	221
Silicosis in Haematite Miners	222
Acute Silicosis	223
Asbestosis	225
<i>Naked Eye Appearances</i>	225
Lungs	226
Mediastinal Glands	228
Other Organs	228
<i>Microscopical Appearances</i>	229
The Asbestos Fibre and the Asbestosis Body	229
The Lungs	239
The Pleura	243
Histology of Other Organs	244

<i>Complications and Sequelae</i>	244
Clinical Pathology and Post-Mortem Examination	246
<i>Sputum</i>	246
<i>Asbestosis</i>	246
<i>Silicosis</i>	247
<i>Blood</i>	247
<i>Urine</i>	248
<i>Autopsies</i>	249
<i>Histology</i>	250
<i>Extraction of Asbestos and Silica Particles from Lung</i>	252
<i>Chemical Analysis of the Lung</i>	253
V. EXPERIMENTAL PATHOLOGY	257
Experimental Pneumoconioses	257
<i>Experimental Methods</i>	260
<i>General Tissue Responses to Various Kinds of Mineral Particles</i>	269
Silicates	271
Free Silica	277

CONTENTS	xv
Amorphous silicas	283
<i>Mixtures of Free Silica and Other Minerals</i>	286
Granite	289
Ferruginous chert	292
Gold	293
<i>Inhalation of Various Dusts</i>	294
Non-siliceous dusts	297
Free silica	301
<i>Inhalation of Mixtures of Free Silica with Other Substances</i>	304
Hematite with 6 per cent Quartz	307
Ferruginous Chert with 50 per cent Silica	309
Artificial Mixtures of Calcined Gypsum and Pure Silica	313
Granite	320
<i>Asbestos</i>	323
<i>Infection and Pneumoconiosis</i>	327